Pediatrics Final 2.0

🚨 **This summary is more comprehensive than the previous version, almost 1.5 times larger, but contains more explanations and is more comprehensive.**

🔊 **Podcast Style Review (Experimental Feature)**

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- **NOTE:** Highlighted in **bold** are the important key info!
- Check the table of contents below for easier navigation
- Good luck 🍀

1. Cardiology

I. Congenital Heart Defects (CHD) & Acquired Conditions

- **Patent Ductus Arteriosus (PDA)**
  - **Large PDA Findings:**
    - Blood pressure example: 100/45 mmHg (wide pulse pressure).
    - **Bounding pulses.**
    - Displaced apical impulse.
    - Left axis deviation on ECG.
    - Cardiomegaly on CXR.
    - Continuous murmur at the left upper sternal border/left subclavicular area.
    - *Not* typically associated with right atrial dilatation on CXR.
  - **Physiology:** Leads to **wide pulse pressure** and prominent (bounding) peripheral pulses due to continuous runoff of blood from aorta to pulmonary artery.
  - **Management (when PDA is vital):** For cyanotic CHDs dependent on PDA for pulmonary or systemic blood flow, **administer Prostaglandin E1 (PGE1)** to keep the ductus arteriosus open.
- **Ventricular Septal Defect (VSD)**
  - **Large VSD Presentation:**
    - Symptoms: Progressive tachypnea, poor feeding, failure to thrive, cool extremities, intercostal retractions.
    - Auscultation: **Holosystolic murmur** on the left sternal border (due to VSD flow), loud second heart sound (P2 component due to pulmonary hypertension).
    - Pathophysiology:
      - Intercostal retractions are due to **pulmonary vascular congestion.**
      - Liver enlargement can occur due to congestive heart failure.
    - ECG/CXR: Left atrial dilatation on ECG, cardiomegaly on CXR.
    - **Symptoms typically start after 4-8 weeks with dyspnea.**
  - **Small VSD:** Often presents with a **loud, high-pitch pansystolic murmur** heard best at the apex or lower left sternal border; the murmur becoming louder can indicate the VSD is getting smaller.
  - Failure to gain weight can be due to increased metabolic demand and poor feeding.
  - *Not* typically associated with wide pulse pressure (unlike PDA).
- **Atrial Septal Defect (ASD)**

- **Clinical Features:**
  - Often asymptomatic.
  - Auscultation: Soft mid-systolic ejection murmur (grade 2/6) at the left upper sternal border (pulmonic area) or left second intercostal space, with **wide and fixed splitting of the second heart sound (S2).**
  - Gallop rhythm is *not* a typical finding of ASD.
- **Complications (Large Secundum ASD):**
  - **Dilatation of the right atrium (RA) and right ventricle (RV).**
  - Risk of tachyarrhythmias (e.g., atrial fibrillation) in adulthood.
  - Risk of paradoxical emboli, increasing with age.
  - Risk of Eisenmenger syndrome is rare and occurs late in uncorrected large defects.
  - Large defects often require closure even if asymptomatic.
- **CXR:** May show enlarged RA and increased pulmonary markings.
- *Not* associated with left ventricular dilatation (causes RV volume overload).

- **Tetralogy of Fallot (TOF)**
  - **Components:**
    1. Ventricular Septal Defect (VSD)
    2. **Pulmonary Stenosis (PS)** (determines severity of cyanosis)
    3. Overriding Aorta
    4. **Right Ventricular Hypertrophy (RVH)**
  - **Clinical Presentation:**
    - **Cyanosis:** Varies with severity of PS; classically presents around 3-9 months.
    - Murmur: **Loud, long, harsh systolic ejection murmur at the left sternal border** (due to RV outflow tract obstruction/PS).
    - **Right ventricular heave** due to RVH.
    - Pulses are typically normal.
    - **Cyanotic spells ("Tet spells"):** Attacks of increased cyanosis, often precipitated by crying, feeding, or defecation.
  - **CXR: Boot-shaped heart** with **decreased pulmonary vascular markings.**
  - **Factors Worsening Cyanosis:** Exercise, fever, dehydration, travel to high altitude (due to decreased SVR or increased PVR).
  - **Factors Improving Cyanosis: Squatting** (increases SVR, promoting pulmonary blood flow).
  - Generally *not* associated with cardiomegaly on CXR unless other complications.
- **Transposition of Great Arteries (TGA)**
  - **Presentation: Early and severe cyanosis in a newborn**, often without a significant murmur unless associated defects (e.g., VSD, PDA) are present.
  - **CXR:** Classic finding is an **"egg-on-a-string" appearance** (narrow mediastinum with ovoid cardiac silhouette).
  - **Survival:** Often dependent on mixing at atrial level (PFO/ASD) or ductal level (PDA).
- **Aortic Stenosis (AS)**
  - **Murmur: Systolic ejection murmur**, typically loudest at the **right upper sternal border**, radiating to the suprasternal notch and carotid arteries.
- **Coarctation of Aorta (CoA)**
  - **Clinical Features:**
    - **Hypertension in upper extremities.**
    - **Radio-femoral pulse delay** and weaker/absent femoral pulses.

        - Displaced apical impulse, LVH on ECG.
        - Systolic murmur often heard best in the inter-scapular area (back).
    - **CXR:**
        - **Rib notching** (due to collateral circulation via intercostal arteries) in older children.
        - Cardiomegaly.
        - *Not* typically associated with a wide mediastinum.
- **Tricuspid Atresia**
    - **Presentation:** Cyanotic CHD.
    - **ECG:** Characteristic finding is **left axis deviation** (due to hypoplastic RV and relatively larger LV).
    - Physical Exam: May have a holosystolic murmur if VSD is present. **No right ventricular heave** (RV is hypoplastic); left ventricle is often enlarged.
- **Hypoplastic Left Heart Syndrome (HLHS)**
    - Survival is dependent on a patent ductus arteriosus for systemic circulation and a PFO for shunting.
- **Truncus Arteriosus**
    - Single arterial trunk arises from the heart, supplying systemic, pulmonary, and coronary circulations. Always associated with a VSD.
    - Leads to volume overload on LV and increased pulmonary vascular markings on CXR.
- **Total Anomalous Pulmonary Venous Return (TAPVR)**
    - Pulmonary veins drain into the right atrium or systemic veins instead of the left atrium.
    - Requires an interatrial communication (PFO/ASD) for survival.
    - CXR: May show “snowman” appearance (supracardiac type).
- **Pulmonary Atresia with Intact Ventricular Septum**
    - Survival dependent on PDA for pulmonary blood flow and PFO for RA to LA shunting.

II. Cardiovascular Physiology & General Principles

- **Fetal Circulation:**
    - **Highest oxygenated blood is in the umbilical vein.**
    - Blood moves from the pulmonary artery to the aorta via the **ductus arteriosus.**
    - The **foramen ovale** allows blood to move from the right atrium to the left atrium.
    - The **right ventricle is dominant** in fetal life, supplying two-thirds of combined ventricular output.
- **Transition to Postnatal Circulation:**
    - Pulmonary vascular resistance (PVR) is high at birth, **decreases significantly in the first few weeks** of life, reaching near-adult levels by 4-8 *weeks*.
    - Foramen ovale functionally closes shortly after birth, anatomically later.
    - Ductus arteriosus constricts and closes, typically within days to weeks.
- **Early Life Cardiovascular Physiology:**
    - **Resting heart rate decreases with age.**
    - **Blood pressure is lowest at birth and gradually increases with age.**
    - Normal BP of a preterm neonate is less than that of a full-term neonate.
    - A heart rate of 165/min in a crying 6-month-old is within normal limits (normal up to ~180/min).
- **Cardiac Function & Output:**
    - During increased metabolic demand, cardiac output increases via increased heart rate and stroke volume.
    - **Low blood pressure is a late sign of decreased cardiac output.**
    - Cardiac output is usually increased in patients with chronic anemia.

    - **Cardiac output *decreases* with increasing afterload.**
    - Sympathetic stimulation increases cardiac contractility.
    - In normal hearts, RV output equals LV output.
    - Cardiac output is determined by stroke volume and heart rate.
- **Volume Overload:**
    - **Left Ventricular Volume Overload:** Caused by PDA, VSD, aortic regurgitation, mitral regurgitation, truncus arteriosus.
    - *Not* caused by ASD (causes RV volume overload) or CoA (causes LV pressure overload).
- **Heart Murmurs:**
    - **Innocent Murmurs:** Common in children. Typically systolic, soft, vibratory, and change with position (e.g., **disappears or diminishes when the child stands up**). Reassurance is often the management.
    - **Diastolic Murmurs: Always pathological.** Causes include aortic regurgitation, pulmonary regurgitation, mitral stenosis, tricuspid stenosis. *Not* coarctation of aorta.
    - Continuous Murmur: e.g., PDA.
- **Cyanosis in CHD:**
    - **Hyperoxia Test:** Used to differentiate cardiac from pulmonary causes of cyanosis. In CHD, arterial PaO2 usually remains **< 150 mmHg** on 100% oxygen.
    - **Management of Unstable Infant with Suspected Cyanotic CHD: Administer Prostaglandin E1 (alprostadil)** to maintain ductal patency.
- **Survival Shunts:**
    - **Patent Foramen Ovale (PFO) dependent lesions:** Tricuspid atresia, TAPVR, HLHS, pulmonary atresia with intact ventricular septum.
    - **Patent Ductus Arteriosus (PDA) dependent lesions:** Pulmonary atresia, critical pulmonary stenosis, critical coarctation/interrupted aortic arch, HLHS, TGA (for mixing). **Tricuspid atresia with no VSD always requires a PDA.**
- **Clubbing:** A sign of chronic hypoxia, typically develops after several months. A 3-month-old is generally too young to show clubbing.

III. Heart Failure

- **Causes in Infancy:** Large VSD, large PDA, CoA, cardiomyopathies.
- **Clinical Signs:** Tachypnea, tachycardia, poor feeding, failure to thrive, hepatomegaly, diaphoresis with feeding, cool extremities, intercostal retractions.
- **Management:**
    - **Diuretics (e.g., Lasix):**
        - Electrolyte disturbances: **Hypokalemia, hyponatremia, hypochloremia, metabolic *alkalosis*, hypercalciuria.**
    - **Inotropes (improve contractility):** Dopamine, epinephrine, digoxin, milrinone.
    - **Beta-blockers (e.g., propranolol):** *Negative* inotropes; contraindicated in acute decompensated heart failure.
    - **Viral Myocarditis with Heart Failure:** Management includes oxygen, diuretics, and inotropic support. **IV fluid bolus and beta-blockers are contraindicated.**
- **Cardiogenic Shock:**
    - Worsening indicators: **Decreased mixed venous oxygen saturation** (e.g., central venous O2 sat from 60% to 45%).
    - Improvement indicators: Decreased serum lactate, increased urine output.

IV. Specific Diagnostic Points

- **ECG in Tricuspid Atresia: Left axis deviation.**
- **Wide and Fixed Splitting of S2: Atrial Septal Defect (ASD).**
- **Boot-Shaped Heart on CXR: Tetralogy of Fallot (TOF).**

- **Egg-on-a-String CXR: Transposition of Great Arteries (TGA).**
- **Rib Notching on CXR: Coarctation of Aorta (in older children).**
- **Systolic Murmur Radiating Suprasternally (Right Upper Sternal Border): Aortic Stenosis.**
- **Opening Systolic Click (Right Side) with Radiation and Systolic Murmur: Pulmonary Stenosis.**
- **Holosystolic Murmur at Left Sternal Border, Loud P2, Cool Extremities, FTT (2 m/o): Ventricular Septal Defect (VSD).**

V. Miscellaneous Cardiovascular Points

- **Syncope/Sudden Loss of Consciousness with Exertion:** Consider aortic stenosis, Long QT syndrome, hypertrophic cardiomyopathy (HOCM).
- **Cardiomyopathy Compensatory Mechanisms:** Increased heart rate, water retention (increased preload), increased sympathetic tone, increased angiotensin II levels. **Polycythemia is a response to chronic hypoxia, not directly a compensatory mechanism for primary pump failure.**
- **Normal Cardiac Findings in 3-Month-Old:** HR 180 when crying, capillary refill 1 sec, visible apical impulse. Louder S2 than S1 at upper sternal border is normal. **BP 110/70 might be high for a 3-month-old.**
- **Umbilical Cord Anomaly (Single Umbilical Artery - one artery, one vein):** Associated with an **increased likelihood of congenital anomalies** (especially renal and cardiac).
- **Asymptomatic Systolic Murmur at LSB that Disappears on Standing: Innocent murmur.**
- **Eisenmenger Syndrome:** A complication of large, uncorrected left-to-right shunts (VSD, ASD, PDA) leading to severe pulmonary hypertension and reversal of the shunt (right-to-left), causing cyanosis. It is a **late complication**, typically in late childhood or adulthood. Risk is lower and later in ASD compared to large VSDs or PDAs.

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2. Respiratory

I. Infections

- **Pneumonia**
  - **Common Causative Pathogens:**
    - **4 months - 4 years: RSV** is a common cause, **Streptococcus pneumoniae** is the most common bacterial cause.
    - **Agammaglobulinemia: S. pneumoniae** is a common cause of both otitis media and pneumonia.
    - **School-aged children (>5 years): Mycoplasma pneumoniae** and Chlamydophila pneumoniae are frequent bacterial pathogens. Mycoplasma is *not* common in early infancy.
  - **Indications for Hospitalization:**
    - **Age < 6 months (not < 6 years).**
    - Signs of **respiratory distress.**
    - **Immune deficiency.**
    - Lobar pneumonia with suspected resistance to beta-lactam antibiotics.
    - **No response to oral antibiotics.**
  - **Mycoplasma Pneumonia:** Drug of choice is a **macrolide (e.g., erythromycin, azithromycin).**
  - **Staphylococcus aureus Pneumonia:** Can be severe, associated with **empyema** and **pneumatoceles** (thin-walled, air-filled cysts on CXR).
  - **Haemophilus influenzae type b (Hib):** Significant cause of lobar pneumonia in unvaccinated children.
  - **Viral Pneumonia:** Viruses are a **common cause of pneumonia in infancy,** especially < 2 years (RSV, parainfluenza, influenza, adenovirus).
- **Bronchiolitis**
  - **MCC: Respiratory Syncytial Virus (RSV).**
  - **Clinical Features:** Tachypnea, wheezing, cough, subcostal/intercostal retractions, nasal flaring.

- **CXR Findings: Bilateral hyperinflation,** atelectasis. *Not* decreased lung volume.
- **Course:** Can take several weeks to resolve, especially cough and wheeze.
- **Complications:** Apnea (especially in young infants/premature), respiratory failure. Secondary bacterial pneumonitis is *not* a common complication.
- **Long-term:** RSV bronchiolitis in infancy may **increase the risk of developing asthma later in life,** but asthma itself is not an acute presentation of RSV.
- **Management:** Supportive. **Nebulized hypertonic saline** may be used. Systemic corticosteroids, routine nebulized bronchodilators, and IV ceftriaxone are generally *not* recommended.

- **Croup (Laryngotracheobronchitis)**
  - **MCC: Parainfluenza virus.**
  - **Clinical Features:** Barking cough, inspiratory stridor, hoarseness. Onset is usually **gradual/insidious**, often preceded by URI symptoms.
  - **Age:** Most common in children **6 months to 3 years.** *Not* more common in children older than 4 years.
  - **Management:** Corticosteroids (oral or nebulized), nebulized epinephrine for moderate/severe cases.
  - **X-ray: Steeple sign** on AP neck X-ray (subglottic narrowing).
  - If symptoms recur after treatment (no drooling, vaccinated): Could be persistence of viral infection or a new viral episode. **Bacterial tracheitis** (often S. aureus) should be considered if high fever, toxicity, and worsening distress develop after several days of croup-like illness.
- **Epiglottitis**
  - **MCC (Historically):** Haemophilus influenzae type b (Hib) - incidence dramatically reduced by Hib vaccine.
  - **Clinical Features: Rapid/sudden onset** (over hours), high fever, drooling, dysphagia, severe sore throat, toxic appearance, **tripod positioning,** inspiratory stridor, muffled voice. Cough is often **absent or slight.**
  - **Diagnosis: Laryngoscopy in a controlled setting (OR)** is definitive. Lateral neck X-ray may show **"thumb sign."**
  - **Management:** Secure airway (intubation in OR), IV antibiotics (e.g., **ceftriaxone**). **Avoid anxiety-provoking interventions** or attempts to visualize pharynx outside controlled setting.
  - **Aminoglycosides are *not* the drug of choice.**
  - Coryza (runny nose) is *not* a typical preceding feature.
- **Bacterial Tracheitis**
  - Serious bacterial infection of trachea, often following viral croup. MCC: **S. aureus.**
  - Presents with high fever, toxicity, stridor, copious purulent secretions.
  - Can be clinically similar to epiglottitis but often has more productive cough.
- **Sinusitis (Acute Bacterial Rhinosinusitis)**
  - **Common predisposing factors:** Viral URI, allergic rhinitis.
  - **Symptoms:** Persistent nasal discharge (often purulent), cough (wet or dry, often worse at night), facial pain/pressure, fever. Thick mucus secretions are a key feature.
  - **Diagnosis:** Primarily clinical. Imaging (CT/plain film) is not routinely needed; normal imaging does *not* exclude diagnosis if clinical suspicion is high. CT is *not* required for diagnosis in uncomplicated cases.
  - **Treatment:** Amoxicillin or amoxicillin-clavulanate is first-line.
  - If unresponsive to amoxicillin (e.g., 10-day course): Consider **antimicrobial-resistant sinusitis** or other causes like **nasal foreign body.**
- **Pharyngitis**
  - **Group A Streptococcus (GAS):** Penicillin remains effective. Treatment decreases risk of **rheumatic fever.**
  - Differentiation between viral and bacterial is often difficult by physical exam alone.
  - Bacterial pharyngitis often has **sudden onset and anterior cervical adenopathy.**
  - Acute glomerulonephritis can follow **streptococcal pharyngitis OR streptococcal skin infection (impetigo).**

II. Non-Infectious Respiratory Conditions

- **Asthma**
  - **Pulmonary Function Tests (PFTs) during exacerbation:**
    - **Decreased FEV1,** FEV1/FVC ratio, PEFR, FEV 25-75%.
    - **Increased Residual Volume (RV),** Functional Residual Capacity (FRC). Total Lung Capacity (TLC) may be normal or increased. *Not* decreased TLC.
  - **Controller Therapy Indication (GINA):** Using SABA (reliever) **more than two days per week.**
  - **Triggers (Extrinsic/Allergic):** Pollen (e.g., olive tree), cat dander, dust mites, molds. Cold air is a non-allergic trigger. **Cow's milk is a food allergen** and can worsen asthma in sensitized individuals, but not a typical inhalant trigger for extrinsic asthma.
  - **Acute Exacerbation:**
    - **Early stages:** Tachypnea leads to respiratory alkalosis (PaCO2 falls).
    - **Severe/Late stages (Impending Respiratory Failure): Hypercarbia (PaCO2 rises)** and respiratory acidosis. PaCO2 of 50 torr is a concerning sign. Silent chest is an ominous sign.
    - **Management (Severe Acute):** SABA nebulization, systemic corticosteroids (e.g., IV hydrocortisone). Inhaled corticosteroids are for long-term control, not primary acute treatment. Magnesium sulfate IV can be used in severe cases.
  - **Predictive Risk of Future Asthma: Skin prick test for inhaled allergens** is a useful investigation.
  - **Most common trigger for asthma in young children (0-4 years): Upper respiratory infections.**
- **Cystic Fibrosis (CF)**
  - **Genetics: Autosomal recessive.** Defect in the CFTR gene (Chloride channel) on **chromosome 7.** Affects males and females equally. Affected patients have two defective CFTR genes.
  - **Diagnosis:**
    - **Sweat Chloride Test:** Gold standard. False negatives can occur with hypoproteinemia. Test results do *not* normalize with treatment. Monitoring sweat chloride is not used to assess adequacy of management. Accurate in newborns.
    - **Genetic Testing:** Confirms diagnosis by identifying CFTR mutations. Does not diagnose 100% of cases as many mutations exist.
    - **Newborn Screening:** Often includes Immunoreactive Trypsinogen (IRT). Elevated IRT is a screening test, requires confirmation.
    - **Early Manifestation: Meconium ileus.**
  - **Pulmonary Manifestations:**
    - Chronic wet cough, recurrent respiratory infections, bronchiectasis, pansinusitis.
    - **Pathogens: Staphylococcus aureus** common early, **Pseudomonas aeruginosa** associated with progressive lung disease.
    - **PFTs:** Typically show an **obstructive pattern** early (decreased FEV1, FEV1/FVC). Restrictive pattern may develop later due to fibrosis.
    - **Complications:** Pneumothorax.
    - Recurrent respiratory infections are primarily due to **thick, viscous mucus and impaired mucociliary clearance,** *not* a primary immune deficiency.
  - **Gastrointestinal Manifestations:**
    - Pancreatic insufficiency leading to **fat malabsorption (steatorrhea)** and protein malabsorption.
    - Failure to thrive, rectal prolapse.
    - **Salt wasting** (especially in hot weather).
    - Cholestasis, biliary cirrhosis.
    - Distal intestinal obstruction syndrome (DIOS).

        - *Not* typically perianal abscess.
    - **Other Manifestations:** CF-related diabetes (insulin-dependent hyperglycemia), delayed puberty (not precocious), nasal polyposis.
    - **Treatment:**
        - Pulmonary therapy (airway clearance), antibiotics for exacerbations (e.g., aminoglycoside + anti-pseudomonal penicillin like ticarcillin for Pseudomonas).
        - Pancreatic enzyme replacement, fat-soluble vitamin supplementation (A, D, E, K).
- **Laryngomalacia**
    - MCC of persistent stridor in infants.
    - **Inspiratory stridor,** often worse when supine, crying, or feeding.
    - Usually **congenital** and improves with age. *Not* very common after surgery.
    - Can be aggravated by GERD.
- **Choanal Atresia**
    - Congenital obstruction of posterior nasal passage.
    - Presents as **neonatal respiratory distress/cyanosis that improves with crying** (as baby mouth-breathes) and worsens with feeding/pacifier use.
- **Respiratory Resistance in Infants:** Major contributor is the **nasal airway and mouth.**
- **Wheezing Causes in Children:** Asthma, cystic fibrosis, viral bronchiolitis, gastroesophageal reflux disease. *Not* lactose intolerance.
- **Transient Tachypnea of the Newborn (TTN)**
    - Benign, self-limiting respiratory distress in term/near-term newborns.
    - Due to **delayed clearance of fetal lung fluid.**
    - Onset usually within hours of birth, resolves by 48-72 hours.
    - More common after Cesarean section.
    - CXR: Perihilar streaking, fluid in fissures, hyperinflation. Air bronchograms are *not* typical (more RDS).
    - Surfactant therapy is **not indicated.**
- **Primary Ciliary Dyskinesia (PCD)**
    - Can present with chronic wet cough, recurrent sinusitis/otitis, bronchiectasis, situs inversus (e.g., dextrocardia).
    - Diagnosis can be aided by electron microscopy of cilia.

III. General Respiratory Points

- **Atypical Pneumonia:** Mycoplasma pneumoniae is a cause.
- **Total Respiratory Resistance in Infants:** Primarily due to **nasal airway and mouth.**
- **Upper Airway Obstruction (Acute):** Causes include croup, laryngitis, bacterial tracheitis, epiglottitis. **Bronchiolitis causes lower airway obstruction.**
- **Normal imaging studies (CT or plain film) of the paranasal sinuses do *not* exclude the diagnosis of acute bacterial rhinosinusitis** if clinical criteria are met.
- **Consolidation (Physical Exam Finding):** Causes **increased tactile vocal fremitus** (not decreased).

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3. Gastrointestinal

I. Infant Nutrition

- **Breast Milk**
    - **Composition:**
        - **Most antigenic part: Protein component.**
        - **Carbohydrate: Lactose** (major component).

        - **Protein: Whey-dominant** (whey:casein ratio higher than cow's milk, e.g., 60:40), easier to digest.
        - **Fat: Long-chain triglycerides,** provides ~50% of calories.
        - **Vitamins/Minerals: Deficient in Vitamin D.** Vitamin K is low (newborns get Vit K shot). Iron content is low but highly bioavailable. Vitamin and mineral content can vary with maternal diet.
        - **Caloric content: Approx. 67 kcal/100 ml.**
        - Amount and composition **vary according to the infant's age** and during a single feed (foremilk/hindmilk).
    - **Benefits:**
        - **Optimal nutrition.**
        - **Easily digestible.**
        - **Reduced risk of infections** (GI, respiratory, otitis media) due to antibodies (IgA), lactoferrin, etc.
        - **Decreased risk of necrotizing enterocolitis (NEC).**
        - **Reduced risk of SIDS.**
        - **Promotes mother-infant bonding.**
        - May decrease risk of asthma, eczema, and obesity.
        - *Does not* inherently protect from hemorrhagic disease of the newborn (requires Vitamin K supplementation at birth).
    - **Breastfeeding Practices:**
        - Initiate immediately after birth.
        - Colostrum is highly beneficial.
        - Feed on demand.
        - **Vitamin D supplementation (400 IU/day) is recommended for all exclusively breastfed infants.**
        - Iron supplementation typically starts at 4-6 months for exclusively breastfed infants.
- **Cow's Milk (Fresh):** Introduction should be delayed until **12 months of age.**
- **Low-Fat Milk:** Introduction recommended at **24 months of age.**
- **Introducing Solid Foods:** Recommended around **6 months of age.**
- **Formula Feeding:**
    - **Soy Formula:** Used for galactosemia, true lactose intolerance (rare), or vegetarian families. Carbohydrate is usually sucrose or corn syrup solids (lactose-free). Contains adequate vitamins if properly formulated. Some cross-reactivity with cow's milk protein allergy (10-15%).
    - **Hydrolyzed Formulas (Extensively or Partially):** Used for cow's milk protein allergy. Amino acid-based formulas for severe allergy.
    - Lactose-free formula is *not* a primary treatment for cow's milk protein allergy (allergy is to protein, not lactose).
- **Evaluation of Adequacy of Nutrition: Consistent and adequate weight gain** is the best indicator. Urine output and head circumference are also monitored.
- **Cow's Milk Protein Allergy (CMPA):**
    - Symptoms: Atopic dermatitis, chronic cough, FTT, vomiting, diarrhea (can be bloody), irritability.
    - Allergy is to **whey and/or casein proteins.**
    - Management (breastfed): Maternal elimination of dairy.
    - Management (formula-fed): Switch to extensively hydrolyzed or amino acid-based formula. Response usually seen within **2-3 days.**

II. Gastrointestinal Disorders

- **Acute Diarrhea & Gastroenteritis**
    - **Causes:** Rotavirus (common), Norovirus, bacteria (Salmonella, Shigella, Campylobacter, E. coli). Amoeba can cause acute diarrhea.

    - **Not a cause of acute diarrhea lasting one week:** Primary lactose intolerance (typically chronic or secondary).
    - **Management:**
        - **Oral Rehydration Solution (ORS) is the cornerstone.**
        - Continue breastfeeding/age-appropriate diet.
        - Avoid high-carbohydrate drinks (can worsen osmotic diarrhea).
        - Antibiotics only if specific bacterial cause identified and indicated (e.g., Shigella). *Not* routinely for EHEC O157:H7 (risk of HUS).
    - **Complications:** Dehydration (most important).
    - Young infants are more severely affected by dehydration. Most cases are self-limited. Specific diagnostic tests usually not needed.
- **Chronic Diarrhea**
    - **Causes:** Giardia lamblia, cystic fibrosis (steatorrhea), lactose intolerance, celiac disease.
    - **Stool pH < 5.5 and presence of reducing substances:** Suggests **carbohydrate malabsorption** (e.g., lactose intolerance), *not* protein malabsorption.
    - **Secretory diarrhea:** Stool output is *not* dependent on oral intake (persists with fasting).
- **Cystic Fibrosis (GI Manifestations):**
    - **Failure to thrive (FTT) due to fat malabsorption** (pancreatic insufficiency).
    - Steatorrhea (bulky, foul-smelling, greasy stools).
- **Celiac Disease**
    - **Immune-mediated enteropathy triggered by gluten** (wheat, barley, rye). Rice is safe.
    - **Presentation:** Diarrhea, FTT, abdominal distension, short stature, irritability. Can present with constipation. Associated with Type 1 DM, autoimmune thyroiditis, Trisomy 21.
    - **Diagnosis:** Serology (Anti-TTG IgA, EMA IgA), small bowel biopsy (gold standard).
    - **Treatment: Lifelong gluten-free diet.** Sensitivity to gluten does *not* resolve.
    - **Complications:** Malabsorption (iron, folate, Vit D, Vit K deficiency - leading to anemia, osteomalacia, bleeding tendency).
    - Increased risk groups: Type 1 DM, Trisomy 21, first-degree relatives. *Not* Trisomy 13.
- **Biliary Atresia:**
    - Presents with neonatal cholestasis (conjugated hyperbilirubinemia), acholic stools, dark urine, FTT.
    - FTT is due to **fat malabsorption** (lack of bile for digestion).
- **Rotavirus Infection:**
    - **Most common cause of severe diarrhea in young children.**
    - Induces **osmotic diarrhea.**
    - Highly contagious.
    - Dehydration is the most important complication.
    - *Not* typically associated with increased stool polymorphonuclear cells (PMNs) unless secondary bacterial infection.
    - **Best diagnostic test: Stool antigen test.**
- **Organic Abdominal Pain Indicators: Pain awakening the patient at night.**
- **Failure to Thrive (FTT):**
    - **Type 1 FTT (Weight <3rd percentile, normal Ht/HC):** Often due to inadequate caloric intake or malabsorption (e.g., incorrect formula prep, persistent diarrhea, vomiting, poor social situation). Chromosomal abnormalities usually cause Type 2 or 3 FTT.
    - **Cystic Fibrosis:** Can cause Type 2 FTT (weight and height affected).

III. Miscellaneous GI Points

- **Hepatitis B Carrier Mother:** Newborn should receive **Hepatitis B vaccine AND Hepatitis B Immune Globulin (HBIG) within 12 hours of birth,** followed by completion of the vaccine series. Breastfeeding is *not* contraindicated if infant receives prophylaxis.
- **Reducing Substances in Urine:** Suggests disorders like **galactosemia.**
- **Bloody Diarrhea:** Caused by Shigella, Salmonella, Campylobacter, EHEC, Entamoeba histolytica.
- **Necrotizing Enterocolitis (NEC):**
  - Most common GI emergency in neonates, especially premature infants.
  - Rate of occurrence inversely related to gestational age.
  - Exact etiology unknown, multifactorial (ischemia, infection, inflammation). Infectious etiology is a contributing factor, not necessarily the sole “most likely causative factor.”
  - Can lead to strictures.
  - Presents with abdominal distension, bloody stools, intestinal perforation (late), hypoactivity, jaundice.

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4. Endocrine

I. Growth & Development

- **Normal Growth Parameters:**
  - **Weight: Doubles by 4-5 months, triples by 1 year.**
  - **Height (Length):** Increases by ~25 cm in the 1st year. **Doubles by approx. 4 years.**
  - **Head Circumference (HC): Increases by ~12 cm in the 1st year.**
  - **Birth Averages:** Weight 2.5-4.2 kg (female avg ~3.2 kg, can be 3.5 kg general), height 48-52 cm.
  - **Upper to Lower Segment Ratio:** At birth ~1.7:1 (torso-dominant). Reaches adult proportions (~0.9-1:1) after puberty.
  - Newborns may lose up to 10% of birth weight in the first week, regained by 10-14 days.
- **Short Stature**
  - **Causes:**
    - **Constitutional Delay of Growth and Puberty (CDGP):** Delayed bone age, normal growth velocity for bone age, family history of similar pattern, eventual normal adult height. More common in boys.
    - **Familial (Genetic) Short Stature:** Normal bone age, normal growth velocity, family history of short stature, short adult height.
    - **Endocrinopathies:** Growth hormone deficiency (GHD), hypothyroidism, Cushing’s syndrome.
    - **Genetic/Chromosomal Syndromes:** Turner syndrome, Down syndrome. Klinefelter syndrome usually results in tall stature.
    - **Systemic Diseases:** Celiac disease, chronic renal disease, IBD.
    - **Skeletal Dysplasias.**
    - **Psychosocial dwarfism.**
    - **Nutritional obesity does *not* cause short stature;** often associated with initially taller stature and advanced bone age.
  - **Evaluation:** Growth velocity, bone age, family history, systemic review. Random GH level is *not* useful (GH is pulsatile); IGF-1/IGFBP-3 are screening tests for GHD.
- **Macrocephaly:** Defined as head circumference **> 97th percentile** for age and sex.

II. Puberty

- **Normal Puberty - Girls:**
  - **First sign: Breast budding (thelarche),** typically between 8-13 years.

- **Menarche:** Usually occurs 2-2.5 years after thelarche, typically around Tanner stage 4. Minimal linear growth occurs after menarche.
- **Peak Height Velocity (PHV):** Occurs around Tanner stage 3, *before* menarche.
- **Delayed Puberty:** No breast development by age 13 or no menarche by age 15/16 (or within 3-5 years of thelarche).
- Onset influenced by genetics (concordance with mother/sisters), ethnicity (earlier in Black girls), nutrition (earlier in obese girls, later in thin athletic girls).

- **Normal Puberty - Boys:**
  - **First sign: Testicular enlargement (≥4 mL volume or >2.5 cm length),** typically between 9-14 years.
  - **Delayed Puberty:** No testicular enlargement by age 14.
  - Peak height velocity occurs later than in girls.
- **Precocious Puberty:** Onset of pubertal signs before age 8 in girls or age 9 in boys.
  - **Central Precocious Puberty:** Early activation of HPG axis. Can be idiopathic or due to CNS pathology (more common to find pathology in boys and very young girls).

III. Thyroid Disorders

- **Congenital Hypothyroidism (CH)**
  - **Most Common Cause: Thyroid dysgenesis** (agenesis, ectopia, hypoplasia) - accounts for ~85% of cases. Usually **sporadic.**
  - **Other Causes:** Dyshormonogenesis (autosomal recessive), TSH deficiency (central), maternal antithyroid drugs (transient), iodine deficiency (endemic).
  - **Epidemiology:** Incidence ~1:3000-4000. Females > males. Less common in Black populations. Screening programs are vital (e.g., TSH, T4).
  - **Clinical Manifestations (Early - often subtle/non-specific in first 4 weeks):**
    - Prolonged jaundice, poor feeding, lethargy, constipation, hoarse cry, large anterior fontanelle, umbilical hernia.
    - Infant may look normal at birth.
    - *Not* typically irritability or excessive crying (more lethargy).
    - *Not* early closure of anterior fontanelle (it's often delayed/large).
  - **Clinical Manifestations (Later/Untreated - fully developed by 3-6 months):**
    - Coarse facial features, large protruded tongue (macroglossia), myxedema (eyelids, external genitalia), short stature, broad hands/short fingers, developmental delay/intellectual disability, hypotonia (not hypertonia).
    - Head size may be normal or slightly smaller if untreated long-term, not always enlarged. Mouth often kept open. Hair coarse.
  - **Diagnosis:** Newborn screening (elevated TSH, low T4/FT4).
  - **Treatment:** Lifelong levothyroxine replacement. **Early treatment is crucial to prevent irreversible intellectual disability.**
- **Central Hypothyroidism:** Low T4 with normal or low TSH. Can be associated with cleft palate.

IV. Adrenal Disorders

- **Congenital Adrenal Hyperplasia (CAH)**
  - **Most Common Form: 21-hydroxylase deficiency** (accounts for >90% of cases).
    - Leads to cortisol and aldosterone deficiency, and androgen excess.
    - **Salt-wasting form:** Presents with hyponatremia, hyperkalemia, dehydration, adrenal crisis in early infancy.
    - **Simple virilizing form:** Androgen excess, normal aldosterone.
    - **Non-classic (late-onset) form:** Milder symptoms.
  - **11-β-hydroxylase deficiency:** Causes **hypertension** (due to accumulation of 11-deoxycorticosterone, a mineralocorticoid), cortisol deficiency, androgen excess.

- **Ambiguous Genitalia:** In 46,XX females due to androgen excess in utero.
- **Diagnosis:** Elevated **17-hydroxyprogesterone (17-OHP)** is key for 21-hydroxylase deficiency. Ultrasound may show uterus in virilized female.
- **Management of Adrenal Crisis:** IV fluids (normal saline), stress doses of hydrocortisone, glucose. Fludrocortisone for mineralocorticoid replacement in salt-wasters (not acutely during stress dose hydrocortisone if that provides enough mineralocorticoid effect).

- **Primary Adrenal Insufficiency (Addison's Disease)**
  - **Lab Findings:** Hyponatremia, hyperkalemia, hypoglycemia, elevated ACTH, positive antibodies to 21-hydroxylase (if autoimmune). **Impaired (low) cortisol response to ACTH stimulation test.**
  - **Clinical Features:** Weakness, fatigue, weight loss, **skin hyperpigmentation** (due to high ACTH), salt craving, postural hypotension.
  - Most common cause of adrenal insufficiency in children overall is **withdrawal of long-term steroids.**
- **Secondary Adrenal Insufficiency:** Due to pituitary ACTH deficiency. **Low ACTH.** Skin hyperpigmentation is absent.
- **Adrenal Crisis (Infant):** Severe dehydration, hyponatremia, hyperkalemia, metabolic acidosis, hypoglycemia, shock. **Renin is high** (due to hypovolemia/hyponatremia). Aldosterone is low (if primary).

V. Diabetes Mellitus & Hypoglycemia

- **Type 1 Diabetes Mellitus (T1DM)**
  - **Most common cause of diabetes in pediatric age group is T1DM (antibody-mediated autoimmune destruction of beta cells).**
  - **Management:** Insulin (long-acting and short-acting), blood glucose monitoring, HbA1c monitoring (target <7.5%), education on diet, exercise, hypoglycemia management.
  - **Screening:** For associated autoimmune conditions like thyroid disease and celiac disease.
  - **Sports:** Children with T1DM **can participate in all sports, including competitive ones,** with appropriate management.
  - This is a **lifelong diagnosis.**
  - **Hypoglycemia Management (Conscious Child, BG 40 mg/dL): Give simple carbohydrates (e.g., cup of juice)**. Glucagon IM/SC if unconscious or unable to take oral.
- **Neonatal Hypoglycemia**
  - **Causes:** Infant of diabetic mother (hyperinsulinism), prematurity, SGA, LGA, perinatal asphyxia, sepsis, adrenal insufficiency, GHD, inborn errors of metabolism.
  - **Congenital hypothyroidism does *not* directly cause neonatal hypoglycemia.**
  - Blood sugar level <2.6 mmol/L (47 mg/dL) in first few hours, then <3.3 mmol/L (60 mg/dL). A level in a 2-hour old may be lower than in a 2-month old without being pathological if transient.
- **Somogyi Phenomenon:** Rebound hyperglycemia following an episode of hypoglycemia (often nocturnal, due to excessive insulin).

VI. Other Endocrine Points

- **Obesity Consequences:** Insulin resistance, Type 2 DM, hepatic steatosis, pseudotumor cerebri, cholelithiasis, sleep apnea, **early menarche** (not late).
- **Disorders Associated with Hypertension:** CAH (11-β-hydroxylase def.), Cushing's, pheochromocytoma, renal artery stenosis.
- **Growth Hormone Deficiency (GHD) in Neonates:**
  - **Clinical Features:** Neonatal jaundice (prolonged), micropenis/microphallus, hypoglycemia, midline facial defects (e.g., cleft palate).
  - **Birth weight is usually normal** as fetal growth is largely GH-independent.
- **Primary Adrenocortical Failure Laboratory Findings:** Elevated ACTH, antibodies to 21-hydroxylase (if autoimmune), hyperkalemia, hyponatremia. A positive (i.e., significant rise in cortisol) high-dose ACTH stimulation test would *rule out* primary adrenal failure; a **blunted or absent response indicates primary failure.** Hypernatremia is *not* a feature.

- **Turner Syndrome:** Associated with congenital heart disease (CoA, bicuspid aortic valve), lymphedema, renal malformations, sensorineural hearing loss, ovarian dysgenesis leading to **short stature and delayed/absent puberty.** Growth velocity is usually *decreased*.
- **Klinefelter Syndrome (47,XXY):** Occurs only in males. Features include tall stature (not short), gynecomastia, small testes, infertility, delayed or incomplete pubertal development.

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5. Neonatology

I. Assessment of the Newborn

- **Gestational Age Definitions:**
  - **Preterm:** < 37 weeks
  - **Early Term:** 37 0/7 - 38 6/7 weeks
  - **Full Term:** 39 0/7 - 40 6/7 weeks (sometimes up to 41 6/7 weeks)
  - **Late Term:** 41 0/7 - 41 6/7 weeks
  - **Post-term: ≥ 42 weeks**
- **Birth Weight Classifications:**
  - **Low Birth Weight (LBW):** < 2500 g
  - **Very Low Birth Weight (VLBW): < 1500 g**
  - **Extremely Low Birth Weight (ELBW): < 1000 g**
  - Small for Gestational Age (SGA): Birth weight < 10th percentile for GA.
  - Large for Gestational Age (LGA): Birth weight > 90th percentile for GA.
- **Apgar Score:**
  - Assesses newborn's adaptation to extrauterine life at **1 and 5 minutes** after birth (and serially if low).
  - Components (0, 1, or 2 points each): **A**ppearance (color), **P**ulse (heart rate), **G**rimace (reflex irritability), **A**ctivity (muscle tone), **R**espiration.
  - **Acrocyanosis (blue hands/feet, pink body) scores 1 for Appearance.**
  - It is a score for **all newborns,** not just term infants.
  - Capillary refill time is *not* part of the Apgar score.
  - It is **used to guide resuscitation efforts,** but decisions about initial resuscitation steps are made before the 1-minute score.
  - It has **limited predictive value for long-term outcomes** like epilepsy or IQ.
  - Crying is *not* a must for score 2 in breathing; regular, strong cry or good respiratory effort scores 2.
- **Physical Examination:**
  - **Initial assessment:** Done immediately after birth by a pediatrician or skilled personnel, especially if risk factors are present.
  - **Full comprehensive examination:** Should be done **within 24 hours of delivery.**
  - Normal cardiac exam at delivery room does *not* rule out congenital heart disease.
  - **Normal findings can include:** Mongolian spots, breast engorgement, pulsating umbilical cord, palpable posterior fontanelle, erythema toxicum, vaginal bleeding/discharge in females.
  - **Abnormal findings can include:** Bifid uvula (may be isolated or syndromic), craniotabes (can be normal in premies or associated with rickets/OI), Wormian bones (can be normal or syndromic), single umbilical artery (associated with anomalies). **Epstein pearls** (small white cysts on palate) and **vernix caseosa** are normal. **Milia** are normal.
  - **Bulging fontanelle is always abnormal.**
  - **One umbilical artery** is abnormal and warrants investigation for other anomalies.
- **Thermoregulation:**
  - Newborns, especially preterms, are at high risk for hypothermia.

- **Management for preterm (e.g., 28-weeker):** Dry immediately, wrap, place in a **radiant warmer or incubator,** use **plastic bag or warming pads.**
- High humidity can *decrease* insensible water losses in incubators.

II. Neonatal Jaundice (Hyperbilirubinemia)

- **Physiological Jaundice:**
  - Appears **after 24 hours of life.**
  - Total serum bilirubin (TSB) rises < 5 mg/dL/day.
  - Peak TSB usually < 12-15 mg/dL in term infants.
  - Predominantly **indirect (unconjugated) bilirubin.** Direct bilirubin < 2 mg/dL or <20% of TSB.
  - Resolves by 1-2 weeks in term infants (may be longer in preterms).
  - **Mechanisms:** Increased bilirubin load (shorter RBC lifespan, higher hematocrit), decreased hepatic uptake, impaired conjugation (immature UGT enzyme), increased enterohepatic circulation.
  - **Exaggerated by:** Prematurity, breastfeeding (due to relative dehydration/caloric restriction initially), low caloric intake, dehydration.
- **Pathological Jaundice:**
  - Jaundice appearing **within the first 24 hours of life.**
  - TSB rising > 5 mg/dL/day or >0.2-0.5 mg/hr.
  - TSB > 15 mg/dL in term infants (thresholds vary with age and risk factors).
  - **Direct (conjugated) bilirubin > 2 mg/dL or >20% of TSB.**
  - Jaundice persisting > 2 weeks in term infants or > 3 weeks in preterm infants.
  - Signs of illness (lethargy, poor feeding, etc.).
  - **Most common cause of jaundice in the first 24 hours of life: Hemolysis** (e.g., ABO/Rh incompatibility, G6PD deficiency, spherocytosis).
- **Breastfeeding Jaundice (Early Onset):**
  - Occurs in the **first week of life.**
  - Due to **insufficient breast milk intake,** leading to dehydration, decreased caloric intake, and increased enterohepatic circulation.
  - Prevented/managed by **frequent and effective nursing.**
- **Breast Milk Jaundice (Late Onset):**
  - Occurs **after the first week of life,** may persist for several weeks/months.
  - Due to substances in breast milk (e.g., β-glucuronidase, pregnanediol) that inhibit bilirubin conjugation or increase enterohepatic circulation.
  - Infant is usually thriving, stools normal.
  - Predominantly indirect bilirubin.
  - High bilirubin levels in breast milk jaundice are generally **not associated with a high risk of neurotoxicity (kernicterus)** if managed appropriately.
  - Usually resolves even if breastfeeding is continued; temporary interruption of breastfeeding is rarely needed.
- **Causes of Unconjugated Hyperbilirubinemia (Prolonged):** Breast milk jaundice, hypothyroidism, Crigler-Najjar syndrome, Gilbert syndrome, pyloric stenosis, ongoing hemolysis. **Urinary tract infection (UTI) can cause conjugated or mixed hyperbilirubinemia,** not typically isolated prolonged unconjugated.
- **Causes of Conjugated (Direct) Hyperbilirubinemia:** Biliary atresia, neonatal hepatitis (idiopathic, infectious - TORCH), choledochal cyst, alpha-1 antitrypsin deficiency, galactosemia, tyrosinemia, cystic fibrosis, Alagille syndrome. **Gilbert syndrome and hypothyroidism cause unconjugated hyperbilirubinemia.**
- **Management of Hyperbilirubinemia:**
  - **Phototherapy:** Converts unconjugated bilirubin to water-soluble isomers.

        - Side effects: Skin rash, dehydration (increased insensible water loss), loose stools, bronze baby syndrome (if direct hyperbilirubinemia present), corneal ulcers (if eyes not protected). **Infection is not a direct side effect.**
    - **Exchange Transfusion:** Indicated for very high TSB levels or signs of acute bilirubin encephalopathy. Factors considered: TSB level, age of neonate (hours/days), presence of hemolysis, risk factors (acidosis, sepsis, prematurity). **Type of feeding (breast vs. formula) is not a primary factor in the decision for exchange transfusion itself, though it influences jaundice development.**
- **Kernicterus (Bilirubin Encephalopathy):** Neurotoxic effects of unconjugated bilirubin deposition in the brain.
- **General Points:**
    - **Visual estimation of jaundice is inaccurate.** TSB/TcB measurement is needed.
    - Normal physical exam does *not* exclude pathological jaundice.
    - Breastfed infants are generally more likely to have hyperbilirubinemia than formula-fed infants.
    - Water supplementation is *not* recommended for breastfed babies to prevent jaundice.
    - IVIG may be used as adjunctive therapy for isoimmune hemolytic jaundice (e.g., ABO/Rh).

III. Respiratory Disorders

- **Respiratory Distress Syndrome (RDS):**
    - **Primary cause: Surfactant deficiency** in premature infants.
    - Most common respiratory disorder of premature infants.
    - **Clinical signs:** Tachypnea, grunting, intercostal/subcostal retractions, nasal flaring, cyanosis. Usually appears within minutes to hours of birth. Breath sounds may be normal or decreased.
    - **CXR: Diffuse symmetrical reticulogranular (ground glass) appearance,** decreased lung inflation, air bronchograms.
    - **Course:** Signs typically peak within 2-3 days (not 5-7 days), then improve with treatment. Worsening after 72 hours suggests complications.
    - **Apnea can be a sign of RDS, but its presence suggests significant disease, not mild.**
    - **Treatment:** Supportive ventilation (CPAP, mechanical ventilation), **exogenous surfactant administration.** Antenatal corticosteroids to mother reduce incidence/severity.
- **Transient Tachypnea of the Newborn (TTN):**
    - Caused by **delayed absorption/clearance of fetal lung fluid.**
    - More common in term/near-term infants, especially after C-section or precipitous delivery.
    - **Onset:** Usually within hours of birth.
    - **Clinical signs:** Tachypnea, mild retractions, occasional grunting.
    - **Course:** Usually self-limiting, resolves within **24-72 hours.** Rarely lasts beyond 48 hours.
    - **CXR:** Perihilar streaking, fluid in fissures, mild hyperinflation. Air bronchogram is *not* a characteristic feature.
    - **Treatment:** Supportive (oxygen if needed). Surfactant is **not indicated.**
- **Meconium Aspiration Syndrome (MAS):**
    - Occurs in term or post-term infants who pass meconium in utero and aspirate it.
    - Meconium-stained amniotic fluid is a sign of potential fetal distress/intrauterine hypoxemia.
    - **Clinical signs:** Respiratory distress, tachypnea, cyanosis.
    - **CXR:** Coarse, patchy infiltrates, hyperinflation, atelectasis. **Air bronchogram is not a primary feature.**
    - **Complications:** Pneumothorax, persistent pulmonary hypertension of the newborn (PPHN).
    - Meconium is sterile, but can cause chemical pneumonitis and predispose to secondary bacterial infection.
- **Grunting Mechanism:** Exhalation against a partially closed glottis to increase end-expiratory pressure and prevent alveolar collapse (auto-PEEP).

IV. Neonatal Sepsis

- **Early Onset Sepsis (EOS):** Occurs **within the first 72 hours of life.**
- **Most Common Pathogens (EOS): Group B Streptococcus (GBS), Escherichia coli.** Listeria monocytogenes. Coagulase-negative staphylococci (CoNS) are more common in late-onset sepsis, especially in VLBW infants with indwelling catheters, and are *not* a typical cause of EOS in term infants.
- **Empirical Treatment (EOS): Ampicillin (covers GBS, Listeria) AND Gentamicin (covers gram-negatives like E. coli).**
- Gram-negative sepsis with meningitis should be treated for at least 2-3 weeks. Neonatal sepsis has high mortality.
- **Most common cause of neonatal sepsis at term: Group B Streptococcus (S. agalactiae).**

V. Other Neonatal Conditions

- **Prematurity Complications:** RDS, apnea of prematurity, osteopenia of prematurity, necrotizing enterocolitis (NEC), intraventricular hemorrhage (IVH), retinopathy of prematurity (ROP), patent ductus arteriosus, sepsis, neurodevelopmental delay, ADHD, lower visual acuity, failure to thrive. **Hypothyroidism is screened for in all newborns; prematurity itself isn't a direct cause but premies are more vulnerable to thyroid dysfunction.**
- **Infant of Diabetic Mother (IDM):**
  - **Complications:** Macrosomia, hypoglycemia (due to fetal hyperinsulinemia), polycythemia, hyperbilirubinemia, RDS (delayed lung maturation), cardiac anomalies (hypertrophic cardiomyopathy, VSD, TGA), hypocalcemia, hypomagnesemia.
  - **Risk of congenital anomalies is *reduced* if maternal diabetes is well-controlled pre-conception and during pregnancy.**
  - Associated with **polyhydramnios,** not oligohydramnios.
- **Postmature Infants (>42 weeks):**
  - **Complications:** Meconium aspiration, PPHN, **hypoglycemia** (due to depleted glycogen stores), polycythemia, birth trauma (if macrosomic), dysmaturity syndrome (peeling skin, long nails, decreased vernix/subcutaneous fat).
  - Often have **chronic intrauterine hypoxia.**
- **Osteopenia of Prematurity:** Diagnosed with labs including **serum alkaline phosphatase and phosphorus.**
- **Apnea of Prematurity:** Caffeine is used to **decrease apnea rate** by stimulating respiratory drive.
- **Cephalohematoma:** Subperiosteal collection of blood, **does not cross suture lines,** confined to one cranial bone. Appears hours to days after birth. Can be a risk factor for jaundice. May calcify. Resolves in weeks to months (not 1 day). Observation is main management.
- **Caput Succedaneum:** Edema of scalp soft tissue, **crosses suture lines,** present at birth, resolves in days.
- **Fluid and Electrolyte Therapy:**
  - **Sodium is generally not added to IV fluids on the first day of life.**
  - Premature infants have higher insensible water losses and need more fluids per kg.
  - **Maintenance fluid for newborns: Glucose 10%** (D10W) or D10 with electrolytes (e.g., D10 ¼ NS) after the first day. Glucose 5% is *not* standard maintenance for neonates.
  - Fluids should be restricted if a hemodynamically significant PDA is present.
  - **Potassium should be added to IV fluids only after adequate urine output is established.**
- **Neonatal Resuscitation:**
  - A skilled person should be present at every delivery.
  - **First 30 seconds are for initial steps:** Warm, dry, stimulate, position airway, suction if needed.
  - Positive pressure ventilation (PPV) if apneic, gasping, or HR <100 bpm. Chest compressions if HR <60 bpm despite adequate PPV.
  - **Epinephrine** (not Atropine) is the drug for bradycardia unresponsive to ventilation/compressions.
  - All newborns are typically placed under a radiant warmer initially for assessment and care; pulse and RR are checked.
- **Infant Botulism:** Prevented by **not giving honey** to infants <1 year old.
- **Single Umbilical Artery:** Associated with increased risk of congenital anomalies (renal, cardiac).

6. Neurology

I. Developmental Milestones & Assessment

- **General Principles:** Development proceeds in a cephalocaudal and proximodistal manner. There's a range for achieving milestones.
- **Gross Motor:**
  - **Head lift (prone):** Present at birth (briefly). Full head support (no head lag when pulled to sit): **4 months.**
  - **Rolls over (prone to supine): 4-6 months.** (Supine to prone usually later, 5-7 months).
  - **Sits with pelvic support:** ~6 months. **Sits without support: 6-8 months.**
  - **Crawls/Creeps:** 8-10 months.
  - **Pulls to stand:** 9-10 months.
  - **Cruises (walks holding onto furniture):** 10-12 months.
  - **Walks with one hand held:** 12-15 months. **Walks alone well (unassisted): 12-15 months** (range 9-17 months).
  - **Runs stiffly: 18 months.** Runs well: 2 years.
  - **Goes upstairs with alternation of feet: 30 months (2.5 years).**
  - **Hops on one foot: 4 years.**
  - **Skips: 5 years.**
- **Fine Motor:**
  - **Tracks visually human faces:** Present at birth/early weeks. **Tracks 180 degrees: 2 months.**
  - **Hands predominantly closed at birth, open by 2-3 months.**
  - **Reaches for objects:** 3-4 months.
  - **Grasps objects (palmar grasp):** 3-5 months.
  - **Transfers objects hand to hand: 5-6 months.**
  - **Immature pincer grasp (thumb and index finger):** 8-10 months. **Neat pincer grasp:** 10-12 months.
  - **Puts objects in mouth: 4-6 months.**
  - **Releases toy on demand:** ~10-12 months.
  - **Scribbles:** 15-18 months.
  - **Draws vertical line: 1.5-2 years. Horizontal line: 2-2.5 years.**
  - **Copies a circle: 3 years.**
  - **Copies a cross: 4 years. Copies a square: 4-4.5 years.**
  - **Copies a triangle: 5 years.**
  - **Cuts picture with scissors: 4 years.**
  - **Ties shoelaces:** 5-6 years.
- **Language:**
  - **Reacts to sounds:** Birth.
  - **Coos (vowel sounds):** 2-3 months.
  - **Laughs: 4 months.**
  - **Babbles (consonant-vowel chains):** 6-9 months.
  - **"Mama/Dada" (non-specific):** 8-10 months. **"Mama/Dada" (specific): 10-12 months.**
  - **Understands "no":** 9-10 months.
  - **Speaks 1-3 words (other than mama/dada): 12 months.**
  - **Speaks 6 words:** ~15-18 months. **10 single words:** ~18 months.
  - **Points to body parts:** 18-24 months.

    - **2-word sentences: 18-24 months.**
    - **Speaks ~50 words:** 2 years.
    - **Knows full name: 30 months (2.5 years).** Knows age: 3 years.
    - **Uses pronouns (me, I, you): 2.5-3 years.** Refers to self by "I": ~3 years (not 4).
    - **Tells stories: 4 years.**
- **Social/Cognitive:**
    - **Social smile: 2 months** (range 1-3 months). Delayed social smile can indicate cognitive problems.
    - **Enjoys looking at mirror:** ~6 months.
    - **Stranger anxiety: 6-9 months.**
    - **Peek-a-boo:** 9-10 months. Waves bye-bye: 9-10 months.
    - **Points to desired object:** 12-15 months.
    - **Knows self age and sex:** Typically by 3-4 years. *Not* at 18 months.
    - **Parallel play:** 2-3 years.
    - **Knows heavier of two objects:** 5 years.
- **Vision:**
    - **Visual fixation:** Present at birth.
    - **Ability to follow a bright target:** Present since birth.
    - **Visual acuity reaches 20/20:** By **3-5 years** (not 6 months).
    - **Ability to distinguish colors:** Develops over first few months; some color vision at birth, good by **5-6 months.** Internet sources vary, but 3 months is plausible for significant development.
- **Developmental Delay:**
    - **Global Developmental Delay (GDD):** Significant delay (≥2 SD below mean) in **two or more** developmental domains (gross motor, fine motor, language, social/personal, cognitive).
    - Most common identifiable cause of GDD is **genetic/chromosomal.** Many cases remain idiopathic.
    - Chromosomal analysis is indicated even without dysmorphic features if GDD is present.
    - **Blind infant:** Prone to **social delay** (due to lack of visual cues for bonding/interaction) and gross motor delay (less motivation for exploration).
    - **Deaf child:** Primarily expected to have **language delay.**
- **Corrected Age for Prematurity:** Use corrected age for developmental assessment until **2 years of age.** (e.g., 7-month-old born at 28 weeks GA = 12 weeks or ~3 months premature; corrected age = 7m - 3m = 4 months).

II. Seizures & Epilepsy

- **Febrile Seizures (FS)**
    - **Simple FS:** Generalized (usually tonic-clonic), duration **< 15 minutes,** does **not recur within 24 hours,** no focal features, child neurologically normal before/after.
    - **Complex FS:** Duration **> 15 minutes,** focal features, and/or **recurs within 24 hours.**
    - **Risk Factors for Recurrence:** Age <18m at first FS, family history of FS, fever <39°C at onset of FS, short duration between fever onset and seizure.
    - **Risk of Subsequent Epilepsy:**
        - Simple FS: Slightly increased risk (2-5%) compared to general population (~1%).
        - **Complex FS, neurodevelopmental abnormalities, family history of epilepsy increase risk significantly.** A simple FS does *not* increase risk by 10 folds.
    - **Management:**
        - Acute: ABCs, abort seizure if >5 min (e.g., rectal diazepam). Antipyretics for comfort.
        - **No need for routine antiepileptic drug (AED) prophylaxis.**

        - **Lumbar Puncture (LP):** Strongly consider if <12 months with first FS. Consider if 12-18 months. If >18 months, based on clinical suspicion of meningitis. Clearly indicated in younger age groups.
- **Neonatal Seizures**
    - **Subtle seizures** are common (e.g., lip smacking, eye deviation, bicycling movements). Change in vital signs can help diagnose.
    - **Treatment of choice: Phenobarbital.**
    - Can carry poor prognosis for neurodevelopment.
    - Causes: Hypoxic-ischemic encephalopathy (HIE), intracranial hemorrhage, infection (meningitis), metabolic (hypoglycemia, hypocalcemia, hypomagnesemia, hyponatremia, hypernatremia), inborn errors, drug withdrawal. **Hypokalemia is *not* a major cause.**
    - Generalized tonic-clonic seizures are *less* common than subtle or focal clonic in neonates.
- **Absence Seizures (Petit Mal)**
    - Brief episodes of staring, loss of awareness, +/- automatisms. **No postictal state.**
    - EEG: **Generalized 3 Hz spike-and-wave discharges.**
    - **Drug of choice: Ethosuximide** (if only absence) or **Valproic acid** (if coexisting GTCs).
- **Partial (Focal) Seizures**
    - **Simple Partial:** Consciousness preserved. Symptoms depend on affected brain area.
    - **Complex Partial:** Consciousness impaired. Often with automatisms. Temporal lobe origin common.
    - **Characteristic of partial complex seizures: Change in level of consciousness.** Epileptic activity often arises from temporal or frontal lobe. Patient usually does *not* remember the event.
    - **Focal seizure with impaired awareness (complex partial):** e.g., child with sudden right arm clonic movement and lack of response.
- **Infantile Spasms (West Syndrome)**
    - Sudden, brief, symmetrical contractions (flexor, extensor, or mixed). Occur in clusters, often on awakening.
    - Onset: Typically 3-12 months.
    - EEG: **Hypsarrhythmia** (chaotic, high-voltage activity).
    - Often associated with underlying brain disorders (e.g., **tuberous sclerosis**).
    - **Treatment: ACTH, Vigabatrin.** Prognosis is often poor for development.
    - Vagal nerve stimulation is *not* first-line treatment.
- **Lennox-Gastaut Syndrome**
    - Severe childhood epilepsy syndrome. Multiple seizure types (tonic, atonic, atypical absence), developmental delay, characteristic slow spike-wave EEG.
    - **Least likely to respond to medical treatment** among common epilepsy syndromes.
- **Rolandic Epilepsy (Benign Rolandic Epilepsy)**
    - Common childhood focal epilepsy. Seizures typically nocturnal, involving face/mouth. Good prognosis.
- **Status Epilepticus**
    - Seizure lasting >5 minutes or recurrent seizures without recovery of consciousness.
    - Associated with ~5% mortality rate.
    - Can cause focal neurological deficits.
    - Intubation is not always required, only if airway/breathing compromised.
- **Gelastic Seizures:** Recurrent attacks of **laughter,** often associated with hypothalamic hamartomas.
- **Differentiation of Seizures vs. Non-Epileptic Paroxysmal Events (e.g., breath-holding spells):** Detailed **history and physical exam** are key. EEG is useful if epilepsy suspected. Breath-holding spells can involve uprolling of eyes, cyanosis, and myoclonic jerks when frustrated/angry/crying.

III. Neurological Conditions

- **Cerebral Palsy (CP)**
  - **Non-progressive disorder of movement and posture** due to an insult to the developing brain. Neurological status (the underlying brain lesion) does not deteriorate, but clinical manifestations can change.
  - **Risk Factors: Prematurity,** low birth weight, intrauterine infections, perinatal asphyxia, multiple gestation. Consanguinity is not a direct risk factor unless it leads to a genetic condition predisposing to CP.
  - **Most common type: Spastic CP.** Diplegic CP is common in preterms.
  - **Choreoathetotic CP:** Often related to **bilirubin encephalopathy (kernicterus)** damaging basal ganglia.
  - **Mental retardation (intellectual disability) is common but not universal.** More common in quadriplegic CP.
  - **The majority of children with CP are born at term,** although prematurity is a major risk factor.
  - Vaccination is *not* contraindicated. Pertussis vaccine is given as per schedule.
  - Language skills do *not* typically deteriorate with time in CP. Deafness does *not* worsen.
  - Tetraplegic CP generally carries a worse prognosis.
  - Epilepsy occurs in about one-third of cases.
- **Acute Ataxia (Child):** If wide-gait, consider post-infectious cerebellitis, intoxication, tumor. **Brain MRI** is an important investigation.
- **Primitive Reflexes:**
  - Assess integrity of brainstem and basal ganglia. Usually symmetrical.
  - **Asymmetry suggests focal brain or peripheral nerve injury.**
  - Most disappear by 4-6 months due to maturation of the frontal lobe.
  - **Persistence beyond expected age or re-emergence can indicate neurological dysfunction.**
  - All primitive reflexes should disappear by 1 year (not 2 years), except protective reflexes.
  - Examples: Moro (disappears 5-6m), Palmar grasp (2-3m), Asymmetric Tonic Neck (6-7m), Rooting (1-2m).
  - **Parachute reflex:** Appears ~8-9 months and **persists for life.**
- **Cerebral Hypotonia:** Can be indicated by other brain abnormalities, fasciculations, ophthalmoplegia. **Hyperreflexia suggests UMN lesion/spasticity,** not hypotonia.
- **Electrolyte Abnormalities Causing Seizures:** Hyponatremia, hypernatremia, hypoglycemia, hypocalcemia, hypomagnesemia. **Hypokalemia is *not* a common direct cause of seizures.**
- **Neurological Examination:**
  - **Vibration sense:** Detected at birth.
  - **Babinski reflex (upgoing toe):** Normal up to **1-2 years.**
  - **Non-sustained clonus (a few beats):** Can be normal up to **2 months.**
  - **Receptive language is usually more mature than expressive language.**
  - **Indirect light reflex:** Present at birth.

IV. Miscellaneous Neurology

- **Valproic Acid Side Effects:** Alopecia, thrombocytopenia, elevated liver enzymes, drowsiness, **weight gain** (not weight loss).
- **Tuberous Sclerosis Complex:** Ash leaf spots, subependymal nodules/calcification, infantile spasms (West syndrome), developmental delay.
- **Sturge-Weber Syndrome:** Port-wine stain (facial nevus flammeus, typically V1 distribution), seizures, glaucoma, developmental delay.
- **Breath-Holding Spells:** Paroxysmal non-epileptic events in young children, often triggered by anger/frustration (cyanotic type) or pain/fright (pallid type). Can involve LOC, cyanosis, brief posturing or clonic jerks. EEG is normal.

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7. Infectious Diseases

I. Vaccinations

- **Live Attenuated Vaccines: MMR (Measles, Mumps, Rubella), OPV (Oral Polio Vaccine), BCG (Bacillus Calmette-Guérin), Varicella, Rotavirus.** Hepatitis A vaccine is an *inactivated* vaccine.
- **Contraindications to DTaP/DTwP:**
  - **Absolute for subsequent doses: Anaphylaxis** to a previous dose or component; **encephalopathy within 7 days** of a previous dose of DTP/DTaP not attributable to another cause.
  - Precautions (defer or consider risk/benefit): Fever ≥40.5°C within 48h; collapse or shock-like state (hypotonic-hyporesponsive episode) within 48h; persistent, inconsolable crying ≥3 hours within 48h; seizure with or without fever within 3 days.
- **Vaccine Not Given at 6 Months (Jordan National Schedule example): Hepatitis A Vaccine (HAV)** (typically given at 12m and 18m). DTaP, OPV, Hib, PCV, IPV are typically given at 6 months in many schedules.
- **Newborn of Hepatitis B Positive Mother: Administer Hepatitis B vaccine AND Hepatitis B Immune Globulin (HBIG) immediately (within 12 hours of birth)** at different sites. Complete the full 3-dose vaccine series (e.g., 0, 1-2, 6 months).
- **Rotavirus Vaccine:**
  - Live oral vaccine.
  - **Contraindicated for children older than 8 months** (first dose not to be given after 15 weeks, series completed by 8 months due to increased risk of intussusception).
  - Effective in decreasing severe diarrhea and dehydration.
  - **Transmitted person-to-person.**
  - *Not* contraindicated in healthy siblings of patients with leukemia (but consult specialist if sibling is actively immunosuppressed).
  - *Not* contraindicated in pregnancy (it's an infant vaccine).
- **MMR Vaccine:**
  - Live attenuated.
  - Previous febrile seizure is *not* a contraindication.
  - Encephalopathy is a very rare adverse effect, but generally considered safe.
  - Varicella can be given at the same time as MMR or separated by at least 4 weeks.
- **OPV (Oral Polio Vaccine):**
  - Live attenuated. Safe in newborns. Recommended in outbreaks.
  - Can cause Vaccine-Associated Paralytic Poliomyelitis (VAPP) in rare cases (incidence much less than 1/1000).
  - IPV (Inactivated Polio Vaccine) is *not* contraindicated after 18 years; it's preferred in many countries to avoid VAPP. OPV is generally not given to adults in non-endemic areas.
  - **OPV is generally superior to IPV in inducing gut immunity and controlling community spread,** but IPV has no risk of VAPP. "Less risk of paralysis" is true for IPV (no VAPP), but OPV is more effective at stopping person-to-person spread.
- **Hepatitis A Vaccine: Killed (inactivated) virus vaccine.** *Not* contraindicated after 12 years (recommended for susceptible individuals).
- **Pneumococcal Vaccine:**
  - Conjugated vaccine (PCV) is indicated for infants <2 years and prevents nasopharyngeal carriage.
  - Polysaccharide vaccine (PPSV23) is poorly immunogenic in children <2 years.
  - *Not* absolutely contraindicated in pregnancy (PPSV23 may be given if high risk). PCV is contraindicated <6 weeks.
  - Pneumococcal vaccine is *not* contraindicated after 5 years; PCV and/or PPSV23 are recommended for high-risk older children/adults.
- **Hib Vaccine (Haemophilus influenzae type b):**
  - Conjugate vaccine. Composed of PRP polysaccharide conjugated to a carrier protein (not outer membrane protein).

    - **Not indicated/needed after 5 years of age** in healthy children.
    - Contraindicated before 6 weeks of age.
    - Prevents Hib carriage.
- **Varicella Zoster Vaccine:** Live attenuated. Generally safe to give to a child with ITP who has been off IVIG for at least **3-11 months** (duration varies by IVIG dose, 9 months is a reasonable specific figure provided).
- **Tetanus Vaccine:** Toxoid. Neonatal tetanus can be prevented by maternal immunization. *Not* contraindicated in immunocompromised individuals (may have suboptimal response). Disease does not confer immunity; vaccine needed after recovery. Tetanus vaccine content (amount of toxoid) *differs* for infants/children (DTaP/DT) vs. adults/older children (Tdap/Td). Composed of inactivated tetanus toxoid.
- **Diphtheria Vaccine:** Toxoid. Adult formulation (Td) contains a smaller amount of diphtheria toxoid than pediatric (DTaP/DT). Not a killed whole cell vaccine.
- **BCG Vaccine:** Live attenuated.
- **General Contraindication to Vaccines: Severe anaphylactic reaction** to a previous dose or to a vaccine component.
- **Vaccination in Immunocompromised Contacts:** Live vaccines (e.g., MMR, Varicella, OPV) are generally **safe to give to close contacts** (e.g., sibling) of immunocompromised individuals (e.g., child with leukemia on chemotherapy), as herd immunity is beneficial. OPV is an exception in some guidelines due to shedding.

II. Specific Infections

- **Varicella (Chickenpox):**
    - Caused by Varicella-Zoster Virus (VZV). Characterized by latency and reactivation as Herpes Zoster (shingles).
    - Outcome is **worse in newborns, immunocompromised individuals, and adults** compared to healthy children. It is *not* milder in adults.
    - Rash typically appears first on the **trunk or face,** then spreads.
    - Vaccine effective post-exposure if given within 3-5 days.
    - Transmission in pregnancy can lead to congenital varicella syndrome or neonatal varicella.
    - Acyclovir is effective for severe infections/complications like encephalitis.
- **Diphtheria:**
    - Immunized individuals can still carry the organism.
    - **Antitoxin should be given as soon as possible** in suspected cases, without waiting for culture confirmation.
    - Vaccine is a **toxoid.**
    - Only toxigenic strains of *Corynebacterium diphtheriae* cause disease.
    - Diphtheria toxin production is phage-induced. Myocarditis is a serious complication.
- **Poliomyelitis:**
    - Neurogical deficit is limited to **motor neurons** (anterior horn cells).
    - Little cross-immunity between the three serotypes (Type 1, 2, 3).
    - **Humans are the only natural host.**
    - Adults have a higher rate of paralysis than children.
    - The overwhelming majority of infections are asymptomatic. Paralysis is usually asymmetrical.
    - Chronic carrier state is rare but described in immunodeficient individuals.
    - Polio is *not* transmitted from cats to man.
- **Hepatitis A:**
    - Rarely acquired by blood transfusion. Most contagious in pre-icteric phase.
    - **Infection is more likely to be severe in adults;** often asymptomatic or mild in young children.
    - Immune globulin (IG) is effective for post-exposure prophylaxis in exposed household contacts.
    - Disease is acquired from humans (fecal-oral).

- **Acute Bacterial Meningitis:**
  - **Poor Prognostic Factors:** Seizure occurring late in course (e.g., on 7th day), focal neurological deficit, Gram-negative organism, development of brain abscess, papilledema at presentation. Convulsions in the first four days *can* affect prognosis.
  - **Empiric Therapy:** Should include a third-generation cephalosporin (e.g., ceftriaxone) and vancomycin.
  - **Dexamethasone:** Should be administered before or with the first dose of antibiotics (within 4 hours of antibiotics) in suspected bacterial meningitis (primarily for Hib and pneumococcal) to reduce neurological sequelae.
  - **Most common in the first five years of life.**
- **Meningococcal Infection (Neisseria meningitidis):**
  - Gram-negative diplococcus.
  - Immunity is type-specific (serogroup-specific).
  - Nasopharyngeal carriage rate is ~5-10% in general population, can be higher.
  - **Prophylaxis for close household contacts is necessary.** (Rifampin, ceftriaxone, or ciprofloxacin). Ceftriaxone is safe for prophylaxis in pregnant women.
  - More severe in patients with **complement deficiency.**
  - **Meningococcemia (sepsis) generally has a worse outcome than isolated meningitis.**
  - Treatment: Penicillin or third-generation cephalosporin (e.g., ceftriaxone). Vancomycin is *not* the drug of choice unless penicillin resistance is high and specific.
  - Low platelet count is a bad prognostic sign.
- **Pneumococcal Disease (Streptococcus pneumoniae):**
  - Most common cause of **occult bacteremia** in children in Jordan (and many places).
  - Only a few serotypes cause the majority of invasive disease.
  - Vancomycin is used for penicillin-resistant pneumococcal meningitis.
  - Most common cause of acute bacterial meningitis in all age groups after the newborn period.
  - Contacts of pneumococcal meningitis generally do *not* need antibiotic prophylaxis (unlike meningococcal or Hib).
- **Pertussis (Whooping Cough):**
  - **Bacteremia rarely occurs.**
  - Vaccine indicated for pregnant women (Tdap) to protect newborns via passive immunity.
  - Acellular pertussis vaccine is generally less effective for long-term protection than whole-cell vaccine but has fewer side effects.
  - Contraindicated in patients with progressive CNS disease.
  - Vaccinating contacts of premature infants is indicated.
  - Highly contagious. Most cases are symptomatic, though can be atypical/milder in adolescents/adults. Patients have lymphocytosis.
  - **Azithromycin is the preferred drug, especially in infants <1 month.**
- **Tetanus:**
  - Caused by toxin produced by *Clostridium tetani*.
  - Disease is toxin-mediated; **bacteremia does not occur.**
  - Patients recovering from tetanus **should receive the tetanus vaccine** as natural infection does not reliably confer immunity.
  - Patients who have received ≥3 doses of tetanus toxoid generally do not require TIG for clean, minor wounds; Td/Tdap booster if >10 years since last dose.
  - Anti-tetanus antibodies (IgG) **do cross the placenta,** providing passive immunity to the neonate if the mother is immunized.
- **Botulism (Infant):** Can be prevented by **not giving honey** to infants under 1 year of age.

- **Mumps:**
  - Transmitted by droplets. **Aseptic meningitis is the most common complication.**
  - Orchitis is more common in post-pubertal males than pre-pubertal boys.
  - Mumps vaccine is *not* contraindicated in thalassemia major (unless other specific contraindications exist).
  - Mumps vaccine can be given to adults if susceptible.
  - Antiviral therapy is of no value.
- **Rubella:**
  - Only one serotype. Vaccine can be given to adults.
  - Primary maternal infection, especially in the **first trimester, poses the highest risk for Congenital Rubella Syndrome (CRS).**
  - Rubella is often **subclinical or mildly apparent,** not almost always clinically apparent.
  - A second dose of rubella vaccine (as part of MMR) is advised for durable immunity.
  - Maximal risk to fetus is in the first two months of pregnancy. Vaccine is contraindicated in patients on high-dose steroids.
  - Congenital Rubella Syndrome is *not* typically characterized by macrocephaly with intracranial calcifications (that's more suggestive of congenital toxoplasmosis or CMV). CRS features: cataracts, heart defects (PDA, PS), sensorineural deafness, microcephaly, blueberry muffin rash.
  - Rash typically starts on the face and spreads caudally.
- **Measles:**
  - Prodrome (cough, coryza, conjunctivitis - "3 Cs") is essential. Highly contagious via respiratory route.
  - Vaccine is *not* contraindicated after 25 years if susceptible.
  - Vaccine can be administered to patients on low-dose/maintenance inhaled steroids; high-dose systemic steroids are a contraindication.
  - Infection is most serious in malnourished and very young children.
  - **Vitamin A supplementation is indicated in all patients** in developing countries or with risk factors, as it reduces morbidity/mortality.
  - Immunity following natural disease is lifelong.
  - Encephalitis occurs in ~1/1000 cases.
  - Contagious by **airborne route** (not just contact).
- **Salmonella Infections:**
  - *S. typhi* (typhoid fever) is primarily human-restricted; acquisition from dogs is unlikely.
  - *S. enteritidis* can be transmitted from contaminated eggs and poultry.
  - Infants develop bacteremia more frequently than older children.
  - Antibiotic treatment of non-typhoidal Salmonella gastroenteritis is generally *not* necessary in immunocompetent older children/adults (can prolong carriage).
  - Invasive Salmonella infections (e.g., osteomyelitis) are more common in patients with sickle cell disease.
- **Post-Exposure Prophylaxis (PEP):**
  - **Hepatitis A:** Hepatitis A vaccine and/or Immune Globulin (IG).
  - **Tetanus:** Tetanus toxoid vaccine and/or Tetanus Immune Globulin (TIG), depending on wound type and immunization history.
  - **Chickenpox:** Varicella vaccine (if given within 3-5 days) or Varicella-Zoster Immune Globulin (VZIG).
  - **Measles:** Measles vaccine (if given within 72 hours) or Immune Globulin (IG).
  - **Diphtheria:** Diphtheria toxoid vaccine and prophylactic antibiotics (e.g., erythromycin). **Antidiphtheric antiserum (antitoxin) is for treatment, not PEP.**
- **Isolation Precautions:**

- Pertussis: Droplet.
- Pulmonary TB: Airborne.
- Rotavirus gastroenteritis: Contact (+ Standard).
- Measles: Airborne.
- **Pneumococcal bacteremia: Standard precautions** (not typically requiring specific isolation unless meningitis with resistant organism or other factors).

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8. Nephrology & Fluids/Electrolytes

Dehydration

- **Assessment:**
  - Mild (3-5%): Slightly decreased intake/output, thirsty, normal exam.
  - Moderate (6-9%): Lethargic/irritable, sunken eyes/fontanelle, decreased tears, dry mucous membranes, decreased skin turgor, prolonged capillary refill (2-3s), tachycardia, decreased urine output.
  - Severe (≥10%): Apathetic/comatose, very sunken eyes/fontanelle, no tears, parched membranes, tenting skin turgor, very prolonged capillary refill (>3s), tachycardia/bradycardia (late), hypotension (late), minimal/no urine output. *Profound signs indicate severe dehydration.*
  - Earliest sign often tachycardia or decreased urine output. Prolonged capillary refill is reliable sign.
- **Types:**
  - Isotonic (most common): Na 130-150 mEq/L.
  - Hypotonic: Na <130 mEq/L. Risk of cerebral edema during rehydration.
  - Hypertonic: Na >150 mEq/L. Doughy skin, irritability/lethargy, increased thirst. Risk of cerebral hemorrhage (due to dehydration) and cerebral edema/seizures if rehydrated too quickly. Correct deficit slowly over 48 hours.
- **Fluid Calculation (Holliday-Segar Maintenance):**
  - 100 mL/kg/day for first 10 kg
  - 50 mL/kg/day for next 10 kg (11-20 kg)
  - 20 mL/kg/day for weight >20 kg
  - Example: 12 kg child = (100×10) + (50×2) = 1000 + 100 = 1100 mL/day.
  - Example: 20 kg child = (100×10) + (50×10) = 1000 + 500 = 1500 mL/day.
  - Example: 25 kg child = 1500 + (20×5) = 1500 + 100 = 1600 mL/day (slide says 1625?).
- **Fluid Management:**
  - Oral Rehydration Therapy (ORT): Preferred for mild-moderate dehydration. Use commercial ORS. Give small frequent amounts. Continue breastfeeding/feeding.
  - Intravenous Fluids (IVF): For severe dehydration, shock, inability to take ORT.
    - Bolus: 20 mL/kg Normal Saline (0.9% NaCl) over 15-30 min (repeat as needed for shock). *7kg child needs 140ml bolus*. *15kg child needs 300ml bolus*.
    - Deficit Replacement: Calculate % dehydration x weight (kg) = deficit (L). Replace deficit + maintenance over 24h (usually half in first 8h, rest over 16h) OR over 48h for hypernatremia.
    - Fluid Choice: Maintenance usually D5 0.45% NaCl or D5 0.2% NaCl (+ 20 mEq/L KCl once voiding). Deficit often replaced with 0.45% or 0.9% NaCl. Initial resuscitation always with isotonic fluid (0.9% NS or LR). *10% dextrose water not appropriate for resuscitation/deficit*. D5W is maintenance fluid for first 24h in newborn without complications.

Urinary Tract Infection (UTI)

- **Incidence:** High in first year, esp. uncircumcised males and females. Prolonged jaundice can be presentation in infants. Constipation is risk factor.
- **Diagnosis:**

- Symptoms: Infants (fever, irritability, vomiting, poor feeding, jaundice), Older children (dysuria, frequency, urgency, incontinence, abdominal/flank pain, fever).
- Urinalysis (Dipstick): Nitrite specific (most Gram-negatives convert nitrate→nitrite), but not sensitive (needs urine in bladder ~4h). Leukocyte esterase (LE) sensitive for pyuria, but not specific for UTI. *Nitrite alone highly sensitive is wrong*. LE+Nitrite most suggestive. Protein/blood non-specific.
- Microscopy: Pyuria (>5 WBCs/hpf). Bacteriuria.
- Culture (Gold Standard): Method matters. Suprapubic aspirate (any growth significant). Catheter specimen (>10^3 - 10^5 CFU/mL significant, depends on source). Clean catch midstream (>10^5 CFU/mL significant). Bag urine unreliable (high contamination rate).

- **Imaging:**
  - Renal Ultrasound (RUS): Done after first febrile UTI in infants/young children to look for structural abnormalities, hydronephrosis.
  - Voiding Cystourethrogram (VCUG/MCUG): To diagnose Vesicoureteral Reflux (VUR). Indications debated, often done after 2nd febrile UTI or first UTI with abnormal RUS/atypical organism/poor response. Not routine after first UTI. *Can detect posterior urethral valves.*
  - DMSA Scan: Detects acute pyelonephritis and renal scarring (gold standard for scarring). Usually done 4-6 months after UTI to assess for scarring. *Should NOT be done in first month to assess scarring*.
- **Management:** Antibiotics (7-14 days for pyelonephritis). Infants <2-3 months usually admitted for IV antibiotics. Older children with pyelonephritis may be treated outpatient if well-appearing/reliable. Treat asymptomatic bacteriuria ONLY in pregnancy (or before urologic procedure).
- **Vesicoureteral Reflux (VUR):** Retrograde flow of urine from bladder to ureters/kidneys. Graded I-V. Lower grades often resolve spontaneously (e.g., Grade II often resolves in 5 yrs). Higher grades associated with renal scarring/recurrent pyelonephritis. Prophylactic antibiotics controversial.

Electrolyte Abnormalities

- **Hyponatremia (Na < 135):**
  - Hypovolemic: Renal losses (diuretics, adrenal insufficiency, RTA) or Extrarenal losses (GI - diarrhea/vomiting, sweat - CF, third spacing). Urine Na low (<20) in extrarenal, high (>20) in renal. *Adrenal insufficiency causes hypovolemic hyponatremia with high urine Na. Gastroenteritis causes hypovolemic hyponatremia with low urine Na.*
  - Euvolemic: SIADH (high urine Na, high urine osm), psychogenic polydipsia (low urine osm), hypothyroidism.
  - Hypervolemic: CHF, nephrotic syndrome, cirrhosis (low urine Na).
  - Cerebral Salt Wasting: Hyponatremia with volume depletion and high urine sodium, usually after CNS injury/surgery.
  - Management: Treat underlying cause. Fluid restriction for SIADH. Normal saline for hypovolemic. Hypertonic saline (3%) for severe symptomatic hyponatremia (seizures) - correct slowly. *Oral salt supplements/steroids not primary tx for SIADH.*
- **Hypernatremia (Na > 145):** Due to water loss > salt loss (DI, fever, burns, diarrhea) or excessive salt intake. See Dehydration section.
- **Hypokalemia (K < 3.5):** Causes: GI losses, diuretics, RTA types 1&2, alkalosis, insulin, beta-agonists. Manifestations: Weakness, fatigue, constipation, paralytic ileus, arrhythmias, ECG changes (U waves, flat T waves). *Confusion less typical.*
- **Hyperkalemia (K > 5.5):** Causes: Renal failure, adrenal insufficiency (Addison's, CAH), acidosis, cell lysis (rhabdo, TLS), K-sparing diuretics, ACEi/ARBs. Manifestations: Weakness, paralysis, arrhythmias, ECG changes (peaked T waves, wide QRS). Management: Calcium gluconate (stabilize membrane), Insulin+glucose, Kayexalate, Bicarbonate, Albuterol, dialysis.
- **Addison's disease (Primary Adrenal Insufficiency):** Causes hypokalemia, hyperkalemia, hyponatremia.
- **SIADH:** Hyponatremia, euvolemic, high urine Na (>40), high urine osmolality (>100), low serum osmolality. Often due to CNS or pulmonary disorders.

Other Nephrology Topics

- **Nephrotic Syndrome:** Characterized by heavy proteinuria (>3.5g/day or >40mg/m2/hr), hypoalbuminemia (<2.5 g/dL), edema, hyperlipidemia. Minimal Change Disease (MCD) most common type in children (responds well to steroids).
  - Features: Edema (scrotal, periorbital, generalized), ascites. *Increased bleeding tendency is NOT typical* (hypercoagulable state due to loss of antithrombin III - low levels expected).
  - Complications: Spontaneous Bacterial Peritonitis (SBP) - often pneumococcal, thrombosis, infections (loss of IgG), AKI (due to hypovolemia or diuretics).
  - Management: Steroids (prednisone) primary treatment for MCD (induces remission in >90%). Salt restriction, diuretics (carefully - risk of hypovolemia). Albumin infusion + Lasix for severe edema/anasarca. ACEi/ARBs for persistent proteinuria. Renal biopsy usually reserved for atypical features (age <1 or >12, hypertension, hematuria, low C3, steroid resistance). *Angiotensin converting enzyme inhibitor is NOT acute management.*
- **Renal Tubular Acidosis (RTA):** Metabolic acidosis with normal anion gap. Associated with FTT, rickets, nephrocalcinosis. Causes hypokalemia (Types 1 & 2) or hyperkalemia (Type 4).
- **Glomerular Filtration Rate (GFR):** Reaches adult levels (around 120 mL/min/1.73m2) by approximately 2 years of age.
- **Asymptomatic Bacteriuria:** Presence of bacteria in urine without symptoms. Generally benign in children, does not require treatment or increase risk of scarring. Can be normal finding in neurogenic bladder.

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9. Miscellaneous

I. Kawasaki Disease (KD)

- **Definition:** An **acute febrile vasculitis, predominantly affecting medium-sized arteries,** of unknown etiology. It is the leading cause of acquired heart disease in children in most developed countries.
- **Diagnostic Criteria:**
  - **Fever persisting for ≥ 5 days** (essential criterion).
  - Plus at least 4 of the 5 following principal clinical features:
    1. **Bilateral non-purulent conjunctival injection.**
    2. **Oral mucous membrane changes:** Erythema and cracking of lips (cracked lips), strawberry tongue, diffuse erythema of oropharyngeal mucosa.
    3. **Extremity changes:**
       - Acute: Erythema and edema of hands and feet (tender swollen hands/feet).
       - Subacute: Periungual desquamation (peeling of skin of fingers/toes).
    4. **Polymorphous rash** (maculopapular, scarlatiniform, or erythema multiforme-like).
    5. **Cervical lymphadenopathy** (usually unilateral, ≥1.5 cm in diameter).
  - Elevated liver enzymes are common but *not* part of the diagnostic criteria. Subcutaneous nodules are *not* a feature of KD.
- **Complications:**
  - **Coronary artery aneurysms (CAA):** Occur in **~20-25% of untreated children.** IVIG treatment reduces this risk to **<5%.** It does *not* occur in the majority of patients if treated.
- **Laboratory Findings:**
  - Elevated ESR, CRP.
  - Leukocytosis with neutrophilia.
  - Normocytic anemia.
  - **Thrombocytosis (high platelet count)** typically develops in the **second to third week of illness.** Thrombocytopenia can occur early in severe cases but is not typical for the course of KD.
- **Treatment:**
  - **Intravenous Immunoglobulin (IVIG):** High dose (2 g/kg) over 10-12 hours.
  - **Aspirin:** High dose initially for anti-inflammatory effect, then low dose for antiplatelet effect.

- **Etiology:** Presumed infectious trigger in genetically predisposed individuals (viral infection is a leading hypothesis but not definitively proven).

II. Malnutrition

- **General Principles:**
  - Severely malnourished patients require **gradual nutritional rehabilitation,** not aggressive full caloric supplementation immediately, to avoid refeeding syndrome.
  - During rehabilitation, serum levels of electrolytes (calcium, magnesium, phosphorus, potassium) must be carefully monitored.
  - Vitamin D and iron deficiency are commonly encountered.
- **Marasmus:**
  - Primarily due to **severe overall caloric (energy) deficiency.**
  - **Most common form of primary protein-energy malnutrition (PEM) globally.**
  - **Clinical features:** Severe wasting (loss of subcutaneous fat and muscle atrophy), emaciated appearance, "old man" facies, alert and irritable. Skin is dry, hair thin and sparse.
  - **Pitting edema is characteristically absent.**
- **Kwashiorkor:**
  - Primarily due to **severe protein deficiency,** often with adequate or near-adequate caloric intake (e.g., starchy diet).
  - **Clinical features: Pitting edema** (starting in lower limbs), dermatosis (flaky paint), hair changes (dyspigmented, sparse), hepatomegaly (fatty liver), apathy, anorexia.

III. Vitamin Deficiencies & Excesses

- **Vitamin A Deficiency:** Night blindness, xerophthalmia, Bitot's spots, keratomalacia, increased susceptibility to infections. **Alopecia is *not* a primary sign** (more related to biotin, zinc, iron deficiency).
- **Vitamin C Deficiency (Scurvy):** Gingivitis, bleeding gums, perifollicular hemorrhage, corkscrew hairs, impaired wound healing.
- **Vitamin D Deficiency (Rickets/Osteomalacia):** Defective bone mineralization.
- **Vitamin E Deficiency:** Hemolytic anemia (especially in premature infants), neurological deficits.
- **Vitamin K Deficiency:** Impaired coagulation, bleeding tendency (e.g., hemorrhagic disease of the newborn).
- **Vitamin B12 Deficiency:** Megaloblastic anemia, neurological symptoms (paresthesias, ataxia, memory loss).
- **Water-Soluble Vitamins:** Vitamin C, B-complex vitamins (B1 Thiamine, B2 Riboflavin, B3 Niacin, B5 Pantothenic acid, B6 Pyridoxine, B7 Biotin, B9 Folate, B12 Cobalamin).
- **Fat-Soluble Vitamins:** Vitamins A, D, E, K. **Vitamin K is fat-soluble.**
- **Terminal Ileum Resection:** Can lead to **Vitamin B12 deficiency** (as B12 is absorbed in the terminal ileum).

IV. Child Abuse

- **Most Frequent Physical Abusers of Children: Parents** (mother alone or both parents together are often cited).
- **Most Common Clinical Manifestation of Physical Child Abuse: Bruises.**

V. Anorexia Nervosa

- **Complications:** Osteoporosis, hypotension, bradycardia (not tachycardia), arrhythmias, constipation, amenorrhea, lanugo hair.

VI. Acrodermatitis Enteropathica

- **Inherited (autosomal recessive) or acquired disorder of zinc absorption/metabolism.**
- Caused by **severe zinc deficiency.**
- **Clinical Features:** Characteristic triad of acral/periorificial dermatitis, alopecia, and diarrhea. Failure to thrive is **usually present,** not absent.

VII. Aspirin Uses in Pediatrics

- **Kawasaki Disease** (anti-inflammatory and antiplatelet).
- **Acute Rheumatic Fever** (anti-inflammatory, though NSAIDs often preferred now).
- *Not* for routine antipyresis in children (Reye's syndrome risk).
- *Not* for thrombocytosis (it's antiplatelet, used to prevent thrombosis, not treat high platelet count itself).
- *Not* for congenital nephrotic syndrome.

VIII. Obesity Complications

- Insulin resistance, Type 2 Diabetes Mellitus.
- Hypertension, dyslipidemia.
- Elevated liver enzymes (Non-alcoholic fatty liver disease - NAFLD).
- Sleep apnea.
- Pseudotumor cerebri.
- **Increased risk of certain malignancies** compared to the normal population (not lower risk).

IX. Sudden Unconsciousness in an Infant

- **Initial Management (First thing to do):** Assess and ensure **Airway, Breathing, Circulation (ABCs).** If airway compromise is suspected (e.g., noisy breathing, drooling), **suctioning the nose and clearing the airway** would be a priority within ABCs. Checking blood sugar is important but secondary to ensuring a patent airway and adequate breathing/circulation.